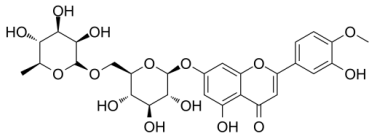
Data Sheet

Product Name:	Diosmin
Cat. No.:	CS-8180
CAS No.:	520-27-4
Molecular Formula:	C28H32O15
Molecular Weight:	608.54
Target:	Aryl Hydrocarbon Receptor
Pathway:	Immunology/Inflammation
Solubility:	DMSO : 32 mg/mL (52.58 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

Diosmin is a flavonoid found in a variety of citrus fruits and also an agonist of the **aryl hydrocarbon receptor** (**AhR**). IC50 & Target: AhR[1] **In Vitro:** Treatment with Diosmin causes a dose dependent increase in the amount of adducts formed (up to a 7-fold increase in adducts at 5 μM Diosmin). At 5 μM, Diosmin increases the cytotoxicity of 7,12-dimethylbenz(a)anthracene (DMBA), shifting the IC_{50} of DMBA from an estimated 1.2 μM to 400 nM. Diosmin is not cytotoxic in itself at the concentrations tested. Diosmin causes an increase in CYPIAI activity in MCF-7 cells in a time- and dose-dependent fashion. Diosmin causes a dose-dependent increase in CYPIAI mRNA after 24 h of incubation, causes a long-lasting increase in CYPIAI mRNA accumu lation that reaches its peak after 48 h of incubation[1]. **In Vivo:** Diosmin significantly decreases the malondialdehyde (MDA) levels and increases the activities of total-superoxide dismutase (T-SOD), glutathione peroxidase (GSH-Px), and catalase (CAT) in the retina of rats compare with the ischemia group ($P<0.05$), and suppresses the ischemia/reperfusion (I/R)-induced reduction in the a- and b-wave amplitudes of the electroretinograms (ERGs) ($P<0.05$). The thickness of the entire retina, inner nuclear layer, inner plexiform layer, and outer retinal layer and the number of cells in the ganglion cell layer are significantly less after I/R injury ($P<0.05$), and Diosmin remarkably ameliorates these changes on retinal morphology. Diosmin also attenuates the I/R-induced loss of retinal ganglion cells (RGCs) of the rat retina ($P<0.05$)[2].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [1]MCF-7 cells are plated at 25,000 cells/well in 24-well plates. After 24 h, the medium is changed to medium containing 5 μM Diosmin. After an additional 24 h, the medium is again changed with medium containing 5 μM Diosmin. After 3 days, the total cell growth is assessed by sulforhodamine[1]. **Animal Administration:** Diosmin is diluted in physiological saline, the vehicle solution.[2] Healthy male Wistar rats (n=112) weighing 180 to 200 g each are used in this study. The animals are randomly assigned to the following 4 groups, which include combinations of the ischemia/reperfusion (I/R) injury model or sham injury with the i.g. administration of Diosmin or vehicle solution: sham+vehicle (SV) group, sham+Diosmin (SD) group, model+vehicle (MV) group, and model+Diosmin (MD) group. For intragastric administration, 5 mL of 2% Diosmin per kilogram weight of the rat, or the same volume of vehicle solution, is administered intragastrically 30 min before the onset of ischemia, and then daily after I/R injury until the animals are sacrificed. Using an overdose of anesthesia, 8 rats from each group are sacrificed 24 h after I/R injury, and their eyeballs harvested for determination of the malondialdehyde (MDA) level and the activities of total-superoxide dismutase (T-SOD), glutathione peroxidase (GSH-Px), and catalase (CAT). At 7 days post-I/R injury, electroretinograms (ERGs) are recorded in 6 rats per group. Meanwhile, 6 rats in each group are randomly chosen for retrograde labeling of retinal ganglion cells (RGCs) , and the remaining 8 rats from each group are histopathologically examined[2].

References:

[1]. Ciolino HP, et al. Diosmin and diosmetin are agonists of the aryl hydrocarbon receptor that differentially affectcytochrome P450 1A1 activity. Cancer Res. 1998 Jul 1;58(13):2754-60.

[2]. Tong N, et al. Diosmin protects rat retina from ischemia/reperfusion injury. J Ocul Pharmacol Ther. 2012 Oct;28(5):459-66.

CAIndexNames:

4H-1-Benzopyran-4-one, 7-[[6-O-(6-deoxy-α-L-mannopyranosyl)-β-D-glucopyranosyl]oxy]-5-hydroxy-2-(3-hydroxy-4-methoxyphenyl)-

SMILES:

O=C1C=C(C2=CC=C(OC)C(O)=C2)OC3=CC(O[C@H]4[C@@H]([C@H]([C@@H]([C@@H](CO[C@H]5[C@@H]([C@@H]([C@H]([C@H](C)O5)O)O)O)O4)O)O)O)=CC(O)=C13

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